Erratum

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MDC1 Directly Binds Phosphorylated Histone H2AX to Regulate Cellular Responses to DNA Double-Strand Breaks

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While shortening our manuscript to the required format, we inadvertently removed reference to the fact that the first evidence for an interaction between MDC1 and γH2AX was provided by Stewart et al. (Nature *421*, 961–969, 2003). This work showed that MDC1 in human cell extracts and MDC1 generated by an in vitro transcription/translation system can be retrieved by an H2AX phosphopeptide but not by an unphosphorylated derivative of this peptide. In addition, we draw attention to the fact that, during the review process of our manuscript, the group of M. Glover published work on the MDC1-H2AX interaction (Lee et al. [2005]. Structure of the BRCT repeat domain of MDC1 and its specificity for the free COOH-terminal end of the γH2AX histone tail. J. Biol. Chem. *280*, 32053–32056. Published online July 27, 2005). The authors showed that the isolated recombinant tandem-BRCT domain of MDC1 can interact directly and specifically with the H2AX C terminus and, on the basis of a crystal structure of the isolated MDC1 twin-BRCT domain, speculated on the nature of the MDC1-γH2AX interaction.